left ventricular dilatation, and thereby prevent or postpone the onset of myocardial disease. Our bias is toward use of digitalis preparations in a patient with left ventricular dilatation, as assessed by clinical, radiographic or echocardiographic techniques, before the onset of either overt depression of systolic ventricular performance or of significant symptoms of heart failure. Certainly, once symptoms of circulatory congestion appear, aggressive therapy with digitalis glycosides, diuretics and sodium restriction is indicated. Again, considering the goal of preventing myocardial dysfunction, systemic arterial hypertension is a particular problem in a patient with aortic regurgitation, because it increases regurgitant volume and systolic wall stress, and the natural history of aortic regurgitation might thereby be accelerated. Therefore, as discussed by Dr. Engler, significant hypertension should be appropriately treated. However, the use of drugs that might decrease the inotropic state of the ventricle or result in bradycardia—such as propranolol, reserpine or guanethedine—should be avoided.

Atrial fibrillation, while uncommon in isolated aortic regurgitation, may be poorly tolerated if atrial systole is an important contributor to end-diastolic volume in a given patient, and cardioversion may be required. Bradyarrhythmias may also be detrimental and may require the use of a permanent pacemaker to reverse the increase in left ventricular volume and pressure caused by long diastolic periods.[4]

Exercise, in general, is well tolerated, as it is accompanied by an increase in heart rate, and a decrease in aortic impedance, both of which reduce the volume of aortic regurgitation.[42] However, any situation which provokes symptoms should be closely evaluated and avoided if it appears to be causing an excessive hemodynamic burden.

Indications for and Objectives of Cardiac Catheterization in Aortic Valve Disease

ALLEN D. JOHNSON, MD

THE TIMING of cardiac catheterization in aortic stenosis, in general, is related to our knowledge of the natural history of this disorder. Once angina, congestive heart failure or otherwise unexplained syncope occurs in a patient with significant aortic stenosis, life expectancy is significantly abbreviated in the absence of surgical relief of the valvular obstruction.[4,21] When any one of these symptoms is present, cardiac catheterization, in anticipation of aortic valve replacement, is indicated. When doubt exists regarding the severity and relative contribution of aortic valve disease in a patient with angina, suspected coronary artery disease, calcium in the region of the aortic valve and a systolic ejection murmur (not an uncommon clinical situation), cardiac catheterization is often necessary to establish the presence and severity of aortic stenosis, and to plan appropriate surgical or medical therapy. More controversial is the decision regarding cardiac catheterization in an asymptomatic patient with suspected severe aortic stenosis. Approximately 5 percent of such patients beyond adolescence will die suddenly without symptoms.[21] However, since operative mortality is in the same range, we prefer to postpone catheterization and surgical therapy until symptoms are present.

The goals of cardiac catheterization in aortic stenosis are:

- *To document the severity of the aortic obstruction.* This requires accurate measurement of the cardiac output simultaneously with measurement of the left ventricular and systemic arterial (preferably ascending aortic) pressures. The aortic valve can usually be crossed retrogradely from the brachial or femoral approach; however, transseptal catheterization of the left ventricle is occasionally required and is particularly useful in such a case. Using the Gorlin formula (aortic valve area = systolic flow/K $\times$ $\sqrt{\text{gradient}}$), the approximate aortic valve area can then be calculated, assuming there is insignificant aortic regurgitation. Careful attention should be directed to the interpretation of the peak and mean systolic aortic valve pressure gradients, as the gradient will vary directly with the square of the systolic blood flow across the valve per unit time, and a low cardiac output could be associated with a low

gradient in the setting of a severely stenotic aortic valve. While "significant" aortic stenosis has been arbitrarily judged to be present if the peak gradient is greater than 50 mm of mercury, and if the aortic valve area index is less than 0.7 sq cm per sq meter, these values should be considered approximate guidelines.[4,21] In a series of symptomatic, medically managed patients with catheterization documented "significant" aortic stenosis by these criteria, neither peak systolic gradient, calculated valve orifice area, cardiac index nor left ventricular end-diastolic pressure separated survivors from nonsurvivors.[4] In general, an aortic valve area less than 0.5 sq cm is considered to represent severe stenosis, and greater than 0.8 sq cm to represent mild stenosis.

• *To document the status of left ventricular function.* The left ventricular angiogram is evaluated qualitatively for wall motion, and quantitatively for ejection fraction, mean systolic ejection rate and Vcf. As Drs. Tsuji and Peterson have discussed, the increased left ventricular mass and wall thickness which accompany compensated aortic stenosis may not be associated with significantly abnormal function of the myocardium at the sarcomere level, and hypertrophy may well regress after relief of aortic obstruction.[12] However, it is well recognized that the late stages of aortic stenosis can be accompanied by severe left ventricular dysfunction with an increased end-diastolic volume and severely depressed ejection fraction. The natural history of unoperated, severe and symptomatic aortic stenosis is so dismal, however, that we exclude no patient from surgical therapy solely because of poor left ventricular function.

• *To document the location of left ventricular outflow obstruction.* While this specialty conference has been limited to valvular disease, it should be recognized that hypertrophic subaortic stenosis can coexist with valvular aortic stenosis,[43] and failure to recognize and document this situation could result in significant intraoperative and postoperative hemodynamic problems. It is important to search for concomitant hypertrophic subaortic stenosis in all cases of valvular aortic stenosis. Echocardiography can be helpful if systolic anterior motion of the anterior mitral leaflet is present. At cardiac catheterization, a positive Brockenbrough response (decrease in systemic arterial pulse pressure on a postpremature ventricular contraction beat), documentation of two levels of systolic pressure gradient (one below the aortic valve and one at the aortic valve) between the left ventricular inflow area and aorta, and high quality biplane left ventricular cineangiograms showing the typical features of anterior position of the anterior mitral leaflet during systole and septal prominence will provide evidence for the presence of hypertrophic obstructive cardiomyopathy.

• *To document the presence or absence of associated valve disease, particularly mitral regurgitation, mitral stenosis and aortic regurgitation.* Simultaneous measurement of pulmonary wedge or left atrial and left ventricular pressures should be done to search for and assess mitral stenosis; left ventriculography should be routinely carried out and will detect mitral regurgitation, and when the presence of significant aortic regurgitation is suspected a supra-aortic valve angiogram should be obtained.

• *To document the status of coronary circulation.* In patients with aortic stenosis beyond the age of 40, the presence or absence of angina pectoris does not accurately predict the presence or absence of coronary artery disease.[44] Since it appears that significant coronary artery disease does influence the operative results in aortic stenosis,[45,46] coronary artery bypass grafting may be warranted if significant coronary artery disease is present.

Aortic regurgitation presents a distinctly different set of problems to a clinical cardiac physiologist. As has been discussed, in chronic aortic regurgitation, myocardial dysfunction usually precedes significant clinical symptoms, and when established may be irreversible.[20] Therefore, if operative mortality and morbidity were negligible, and prosthetic valves infinitely durable and free from problems of thromboembolism, infection and hemolysis, aortic valve replacement could be timed to prevent the establishment of myocardial dysfunction. Further complicating the management of patients with aortic regurgitation is the lack of established clinical criteria, including noninvasive methods, for determining the presence of abnormal myocardial function. Our current approach to the timing of cardiac catheterization and valve replacement in patients with aortic regurgitation takes each of these factors into consideration. In all patients with symptoms of mild to moderate congestive left heart failure, in whom clinical and noninvasive data support the diagnosis of significant aortic regurgitation, catheterization studies are done. If significant depression of left

ventricular performance is present, surgical therapy is recommended even for class II (New York Heart Association) patients. In asymptomatic patients in whom there is evidence of progressive left ventricular enlargement on physical examination, chest x-ray or echocardiogram, or in whom electrocardiograms show increasing signs of left ventricular hypertrophy, or in patients in whom echocardiographic indices of left ventricular performance indicate a progression from normal to abnormal left ventricular systolic performance, cardiac catheterization is done. If left ventricular systolic function as assessed at catheterization is normal, careful medical management is recommended. The presence of significant depression of left ventricular performance warrants consideration for valve replacement. It is worth emphasizing that symptoms of congestive heart failure in patients with chronic aortic regurgitation are not as valuable in predicting survival following valve replacement as other measures of cardiac performance.[47]

A vexing problem encountered in patients with chronic aortic regurgitation is management of a patient who has significant aortic regurgitation and established, *severe* myocardial dysfunction. The natural history in this setting is poor, with progression of myocardial disease and limited longevity.[5,6] However, valve replacement in such cases does not appear to result in the reversal of myocardial dysfunction[20] and in preliminary studies the long-term results of surgical operation in this situation have not been encouraging.[48] In summary, one must decide between medical management with almost certain worsening of myocardial function, and the risk of surgical operation with the possibility of arrest without reversal of the deterioration of myocardial function, and no current evidence that the natural history will be positively affected. If symptoms are refractory to optimum medical management, valve replacement can be undertaken with the above mentioned expectations kept in mind.

At present, cardiac catheterization provides the most reliable means of assessing the function of the left ventricle in patients with aortic regurgitation; therefore, appropriate care and attention should be directed toward the assessment of left ventricular performance at the time of catheterization in these patients. Ideally, quantitative biplane ventriculography should be employed in patients with chronic aortic regurgitation. A recent study by Bolen and associates identified a group of patients with aortic regurgitation and normal basal left ventricular ejection fraction who responded to afterload stress (angiotensin) with a decrease in ejection fraction and left ventricular stroke work index. The authors felt that this response might indicate a state of latent left ventricular dysfunction.[49] Employment of a well-defined stress of this type might prove to be useful in the routine evaluation of left ventricular performance in aortic regurgitation.

Supravalve aortic root angiography is employed to roughly quantitate the degree of aortic regurgitation (trivial to 4+) and to document the anatomy of the aortic root, which may be quite dilated in Marfan syndrome or cystic medial necrosis. Coronary angiograms are routinely obtained in patients over age 40. Associated mitral valve disease—rheumatic and mitral prolapse in particular—should be evaluated with appropriate pressure recordings and analysis of the left ventricular cineangiogram.

Acute aortic regurgitation may overwhelm the usual compensatory mechanisms accompanying a volume overload, and left ventricular end-diastolic pressure may rise to produce severe pulmonary vascular congestion. If medical therapy, as outlined by Dr. Karliner, is not effective in relieving the congestive heart failure, cardiac catheterization followed by aortic valve replacement may be required, even in patients with active infective endocarditis. In patients with acute aortic regurgitation, therefore, the timing of cardiac catheterization and aortic valve replacement is dependent upon the severity of associated signs and symptoms of heart failure, in contrast to chronic aortic regurgitation.

Surgical Considerations in Aortic Valve Disease

PAT O. DAILY, MD*

As Dr. Johnson has discussed, the timing of aortic valve surgical therapy in a given patient requires synthesis and consideration of all the available clinical, noninvasive, angiographic and hemodynamic information. Presented with a patient in whom the decision for valve replacement has been made, a surgeon is faced with two prob-

*Associate Professor of Surgery.